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2004 Annual Report

## Addressing Unmet Cardiovascular Needs

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FINANCIAL

Myogen® 

	Potential Indication	Pre-clinical	Phase I	Phase II	Phase III	Marketed
Perfan® I.V. (i.v. enoximone)	Acute Decompensated Heart Failure					
Enoximone Capsules	Advanced Chronic Heart Failure					
Ambrisentan	Pulmonary Arterial Hypertension					
Darusentan	Resistant Systolic Hypertension					
Drug Discovery	Heart Failure	Research				

## Cardiovascular Disease



The term cardiovascular disease is used to describe a continuum of clinical conditions resulting primarily from three underlying chronic diseases: atherosclerosis, hypertension and diabetes. These underlying diseases cause permanent damage to the heart, blood vessels and kidneys, leading to progressively debilitating clinical conditions such as chronic heart failure, angina, heart attack, stroke, systolic hypertension and chronic kidney disease.

Cardiovascular disease is the leading cause of death and disability in the United States, accounting for 19% of all hospitalizations and over 60% of total mortality in 2002. The American Heart Association estimates that the total direct and indirect costs of cardiovascular disease in the United States will be approximately \$394 billion in 2005, including \$46 billion in costs for drugs and related medical durables and \$140 billion in hospitalization and nursing home costs. Despite improved treatments and increased awareness of preventive measures, approximately 70 million people in the United States currently are living with one or more types of cardiovascular disease.

## Corporate Profile



Myogen is a biopharmaceutical company focused on the discovery, development and commercialization of small molecule therapeutics for the treatment of cardiovascular disorders. Myogen currently markets one product, Perfan I.V.<sup>®</sup>, in Europe for the treatment of acute decompensated heart failure and has three oral product candidates in late-stage clinical development: enoximone for the treatment of patients with advanced chronic heart failure, ambrisentan for the treatment of patients with pulmonary arterial hypertension and darusentan for the treatment of patients with resistant systolic hypertension. Myogen, in partnership with Novartis, also conducts a comprehensive drug discovery research program focused on the development of disease-modifying drugs for the treatment of chronic heart failure and related cardiovascular disorders.

## 2004 Accomplishments



- Initiated ARIES-1 & ARIES-2, the two pivotal Phase 3 trials evaluating ambrisentan in patients with pulmonary arterial hypertension (PAH).
- Completed EMOTE, a non-pivotal Phase 3 trial of enoximone capsules in 201 patients with advanced chronic heart failure, the preliminary results of which were reported in March, with detailed results presented at the annual meeting of the Heart Failure Society of America in September.
- Detailed results of the Phase 2 trial of ambrisentan in 64 patients with PAH presented at the American Thoracic Society annual meeting.
- Completed enrollment and drug treatment of 1,854 patients in ESSENTIAL I & II, the two pivotal Phase 3 trials evaluating enoximone capsules in patients with advanced chronic heart failure.
- Initiated the Phase 2b trial of darusentan in patients with resistant systolic hypertension.
- The U.S. Food and Drug Administration (FDA) granted ambrisentan orphan drug designation for the treatment of PAH.
- Completed a \$60 million financing.

## Addressing Unmet Cardiovascular Needs...



Cardiovascular diseases are the number one killer of men and women. In the United States, cardiovascular diseases claim one victim every 34 seconds.

### To Our Shareholders:

Cardiovascular diseases claim more lives each year than the next five leading causes of death combined. The cost in human life is immeasurable, as is the impact on patients' families. The cost to our medical system, however, is measurable. In 2004, the estimated direct and indirect cost of cardiovascular disease was \$368.4 billion. By comparison, in 2003, the cost of all cancers was \$189 billion. Despite significant progress in past decades, there clearly remain significant unmet needs to be addressed in the treatment of cardiovascular diseases.

Myogen was founded in 1996 with a mission to improve the treatment of cardiovascular disease. Many current therapies for cardiovascular disease do not adequately address the underlying molecular mechanisms of the disorders that make up the diseases. Our understanding of the biology of

cardiovascular disease combined with our clinical development expertise has guided a strategy with two mutually supportive initiatives:

- Selectively in-license and acquire drugs in clinical development for which we have a unique ability to add value, and
- Leverage our understanding of cardiovascular disease at the molecular level in the discovery and development of disease modifying therapeutics.

We have made significant progress under this strategy. We have in-licensed three separate compounds that are in late-stage clinical evaluation for three separate cardiovascular indications. We also have a drug discovery partnership with Novartis based on Myogen's proprietary drug targets and lead compounds and focused on the discovery, development and commercialization

of new therapeutics for the treatment of heart muscle disease.

2004 was a productive and challenging year. We made significant progress in the development of our three product candidates—**enoximone**, **ambrisentan** and **darusentan**—as well as in our discovery research program. At the beginning of 2004, we announced five major corporate milestones for the year, all of which were achieved (see page one). An additional highlight was the completion of a \$60 million financing in September. Despite our progress in 2004, the financial markets did not view these accomplishments positively, as is reflected in our valuation decline during 2004.

However, our achievements last year have positioned Myogen for an exciting year in 2005 in which we expect to obtain results from four clinical trials. This year, we will learn how far we have progressed with Myogen's mission and what impact we may have on three significant cardiovascular disorders.

**Enoximone** We are evaluating the chronic administration of enoximone capsules as a treatment for patients with advanced heart failure. ESSENTIAL I & ESSENTIAL II are our two pivotal Phase 3 trials of enoximone capsules in patients with Class III and IV chronic heart failure. We enrolled 1,854 patients in the trials between February 2002 and May 2004. Patient treatment was completed in November of last year with a mean treatment period for patients exceeding 18 months. All final patient visits were completed by January 2005. Collection of the trial data is progressing according to plan and we expect to report preliminary results of the trials in the middle of 2005.

**Ambrisentan** At the beginning of 2004, we announced the initiation of patient enrollment in ARIES-1 & ARIES-2, the two pivotal Phase 3 trials evaluating ambrisentan in patients with pulmonary arterial hypertension. We expect to complete patient enrollment in ARIES-2 by the

end of June 2005 and ARIES-1 in the fourth quarter of 2005. We plan to report preliminary results from each of the ARIES trials approximately six months following the completion of enrollment in each trial.

**Darusentan** In July 2004, we initiated a Phase 2b randomized, double-blind, placebo-controlled 10-week clinical trial to evaluate the safety and efficacy of darusentan in patients with resistant systolic hypertension. We expect to complete the trial in the middle of 2005 and report trial results one to two months thereafter.

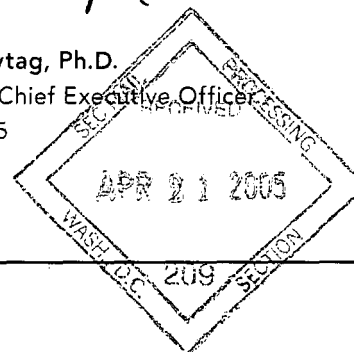
If our clinical programs are successful and our product candidates receive regulatory approval, each of these product candidates has the potential to improve the treatment options available to the patients with these diseases.

These improvements would represent only a step in the right direction for reducing the severity and pervasiveness of cardiovascular diseases. There is more work to be done. Declines in death rates from cardiovascular diseases are largely responsible for the recent major improvements in life expectancy. According to the Centers for Disease Control and Prevention/National Center for Health Statistics, if all forms of major cardiovascular disease were eliminated, life expectancy would rise by almost seven years. Since Myogen's founding eight years ago, we have worked to build an organization that was responsible for improving the quality of life of patients who suffer from debilitating cardiovascular disorders. In 2005, we will learn the results of our efforts thus far.

I look forward to sharing our results with you during the year. Thank you for your continued support.



J. William Freytag, Ph.D.  
President and Chief Executive Officer  
March 25, 2005

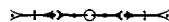




## Chronic Heart Failure

In the United States, approximately five million patients live with chronic heart failure, with an additional 550,000 new cases reported each year. It is estimated that half of all individuals with chronic heart failure die within five years of diagnosis. Chronic heart failure is one of the largest health problems in the developed world, with annual direct and indirect healthcare costs in the U.S. alone exceeding \$28 billion.

## Chronic Heart Failure



Chronic heart failure, also referred to as congestive heart failure, is a debilitating condition that occurs as a damaged heart becomes progressively less able to pump an adequate supply of blood throughout the body. This results in patients developing progressive symptoms of fatigue, shortness of breath (dyspnea), exercise limitation and fluid accumulation (edema). As the severity of chronic heart failure increases, patients develop worsening symptoms and acute decompensation resulting in periodic hospitalization, intensification of therapy and death.

Chronic heart failure has several causes. It generally develops in individuals with a long history of poorly controlled high blood pressure, coronary artery disease or in those who have suffered some other heart-damaging event.

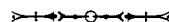
Although medical therapy is improving, heart failure remains a major debilitating and progressive condition characterized by high mortality, frequent hospitalization and deteriorating quality of life. The severity of chronic heart failure is typically classified using a system established by the New York Heart Association that assesses the patient's degree of functional limitation based primarily on shortness of breath. This system is divided into four classes, I through IV, with Class IV being the most severe. Physicians use this

system to track patients' disease progression and responses to therapies.

Following diagnosis, patients with chronic heart failure are typically treated with multiple oral medications, including diuretics, digoxin, vasodilators, angiotensin converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs) and beta-blockers. Diuretics reduce the fluid overload and edema. Digoxin acts to increase the force of cardiac contraction (inotropy). ACEIs and ARBs inhibit the renin-angiotensin-aldosterone hormone system (RAAS). Inhibition of the RAAS system has proven an important advance in the treatment of chronic heart failure, particularly the early stages, and has been demonstrated to improve patient symptoms and survival. Beta-blockers suppress the stress placed on the heart by increased levels of the hormones angiotensin and norepinephrine and have demonstrated an ability to increase patient survival time.

Despite these important advances, over the last few years, clinical studies have demonstrated that further inhibition of the neurohormonal system with additional investigational drugs had not resulted in patient benefit. Thus there is a significant unmet medical need for therapies to treat the "pump failure" that typifies advanced chronic heart failure.

## What Is Myogen Doing?

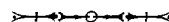


We believe that individuals with advanced heart failure can benefit greatly from the availability of a product that can be used chronically on an outpatient basis to provide symptomatic relief, improve quality of life and reduce the frequency of hospitalizations by delaying episodes of acute decompensated heart failure.

Enoximone, a positive inotrope, is a small molecule that inhibits type-3 phosphodiesterase (PDE-3), an enzyme that is present in the heart and plays an important regulatory role in cardiac function. Enoximone blocks the action of this enzyme, increasing the force of contraction of the heart, thereby

increasing cardiac output. Enoximone also produces vasodilation and enhances ventricular filling, thereby further increasing cardiac efficiency. We are currently conducting Phase 3 clinical evaluation of low-dose oral enoximone in patients with advanced chronic heart failure. We plan to report preliminary results for the pivotal trials, ESSENTIAL I & II, in the middle of 2005. If our clinical program is successful and we receive regulatory approval, enoximone capsules would be the first oral inhibitor of PDE-3 to be commercialized for the treatment of advanced chronic heart failure.

## A Word from the Physician



"Heart failure used to be viewed as a relentlessly progressive disease associated with substantial disability and early mortality in all patients. With improved current and future therapies, heart failure may now be seen as a chronic disease syndrome associated with improved quality of life and longevity in most patients."

—William T. Abraham, M.D.,

Professor of Medicine, Chief, Division of Cardiovascular Medicine, The Ohio State University, Columbus, Ohio

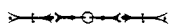


## **Chronic Heart Failure: Research and Development**

Patients with chronic heart failure develop an enlargement of the heart called cardiac hypertrophy. The causes and effects of cardiac hypertrophy have been extensively documented, but the underlying molecular control mechanisms, or cardiac signaling pathways, remain poorly understood.



## Understanding the Molecular Basis of Heart Failure

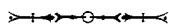


We believe that the fundamental drivers of pathological remodeling of the heart (abnormal growth, shape and function of the heart) are increases in ventricular wall stress and neurohormonal and growth factor stimulation of cardiac muscle. These processes are set in motion by primary insults to the heart, including myocardial infarction (heart attack) and chronic high blood pressure. Wall stress and associated growth promoting stimuli lead to changes in heart

muscle signaling pathways that ultimately produce pathological changes in gene expression in the heart.

One of the characteristic changes that occurs in a failing heart is a change in gene expression wherein fetal genes that were turned off shortly after birth are reactivated in the disease process. Although this response may initially be beneficial to a patient with chronic heart failure, it becomes harmful as the disease progresses.

### What Is Myogen Doing?



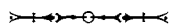
Our scientists and academic collaborators affiliated with the University of Colorado and the University of Texas Southwestern Medical Center are focused on elucidating the underlying molecular mechanisms important in the progression of heart failure. Work conducted in Colorado in the laboratory of Dr. Michael Bristow, one of our founders and our Chief Medical Officer, is focused on identifying the set of fetal genes that are reactivated in chronic heart failure, understanding the consequences of their reactivation and discovering the means to control their expression. This work has led to the discovery of what we believe to be an important gene reactivation that occurs in the failing human heart, that appears to be responsible for weakening the contraction of the heart. Understanding cardiac signaling pathways is a central theme of the research of another scientific collaborator, Dr. Eric Olson at the University of Texas Southwestern Medical Center. This work has led to the discovery of several signaling pathways that appear to control cardiac hypertrophy.

An essential component of our drug discovery strategy is to target the elements of gene expression regulation in the heart that are common to known cardiac remodeling and heart failure pathways. Of primary interest in this regard are the calcineurin, NFAT and

MEF2 signaling pathways and their regulation by Class II histone deacetylases (HDACs), enzymes that repress gene transcription, and other regulatory proteins. NFAT is a transcription factor (controls gene expression) that is regulated by the enzyme calcineurin in the heart and other tissues. MEF2 is a transcription factor regulated by Class II HDACs. In addition, we, together with our collaborators, have discovered what we believe to be an important pathological role for Class I HDACs in pathological cardiac remodeling, and we have patented the use of HDAC inhibitors for treatment and prevention of cardiac disease.

We have developed a series of high-throughput screening assays based on these discoveries and have identified several lead compounds that appear to inhibit cardiomyocyte hypertrophy and/or reverse abnormal fetal gene expression. These compounds are currently being studied in our laboratories in cell and animal assays to examine safety and efficacy and optimization of lead structures is underway. In October 2003, we established a research collaboration with the Novartis Institutes for BioMedical Research, Inc. to augment and accelerate the discovery of compounds that target the fundamental causes in chronic heart failure.

### A Word from the Scientist



"Cardiac remodeling is associated with the activation of a pathological gene program that weakens cardiac performance. Thus, targeting the disease process at the level of gene expression represents a potentially powerful therapeutic approach. An understanding of the mechanistic underpinnings of heart failure represents an essential step toward developing novel therapeutics that will improve the quality of life and prolong survival of heart failure patients."

—Eric N. Olson,

*Professor and Chairman, Department of Molecular Biology, University of Texas Southwestern*



## **Pulmonary Arterial Hypertension**

Approximately 60,000 individuals in the United States and 100,000 individuals in Europe live with pulmonary arterial hypertension (PAH), a highly debilitating disease of the lungs. The only known cure for PAH is lung transplantation. Despite recent advances in therapeutic options that have provided some symptomatic relief and improved survival, there remains an unmet need for additional pharmacologic alternatives for the treatment of PAH.

## Pulmonary Arterial Hypertension

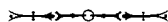


Pulmonary arterial hypertension (PAH) is a progressive and life-threatening lung condition characterized by increased pulmonary artery pressure and pulmonary vascular resistance leading to right ventricular heart failure and eventually death, if untreated. PAH can occur with no known underlying cause which is referred to as idiopathic PAH, or can be associated with connective tissue diseases like scleroderma, congenital heart defects, HIV infection, and other conditions. Patients with PAH develop symptoms of progressive fatigue, shortness of breath, edema and exertion limitation as the heart struggles to pump against the high pulmonary pressure.

Historically, treatment has consisted of diuretics, digoxin and anticoagulants, and calcium channel blockers in select patients. As patients progress into more advanced stages of PAH, therapeutic options include continuous intravenous or subcutaneous infusion of prostacyclin, or more recently, inhaled prostacyclins. In 2002, an important advance occurred with the approval of an orally administered dual-selective endothelin receptor antagonist (ERA).

Endothelin is a small peptide hormone that is believed to play a critical role in the control of blood flow and cell growth. Elevated endothelin blood levels are associated with several cardiovascular disease conditions, including pulmonary hypertension, chronic kidney disease, coronary artery disease, hypertension and chronic heart failure. Therefore, it is believed that agents that block the detrimental effects of endothelin may provide significant benefits in the treatment of some of these conditions. There are two classes of endothelin receptors,  $ET_A$  and  $ET_B$ , which play significantly different roles in regulating blood vessel diameter. The binding of endothelin to  $ET_A$  receptors located on vascular smooth muscle cells stimulates vasoconstriction, cell proliferation and hypertrophy. However, the binding of endothelin to  $ET_B$  receptors located on the vascular endothelium causes vasodilation through the production of nitric oxide and prostacyclin. The activity of the  $ET_B$  receptor is thought to be counter-regulatory, protecting against excessive vasoconstriction and proliferation, and is involved in the clearance of endothelin.

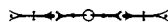
## What Is Myogen Doing?



We believe that a significant opportunity exists for a new class of ERAs that bind selectively to the  $ET_A$  receptor in preference to the  $ET_B$  receptor. Selective  $ET_A$  antagonists are likely to block the negative effects of endothelin by preventing the harmful effects of vasoconstriction and cell proliferation, while preserving the beneficial effects of the  $ET_B$  receptor. Ambrisentan is an oral  $ET_A$  selective ERA. We are currently conducting two Phase 3 clinical trials of ambrisentan in patients with PAH. We believe the selectivity

and potency of ambrisentan may offer significant advantages over other ERAs, including enhanced and more durable efficacy, improved safety, once daily dosing and ease of use (alone or in combination with other therapies), all of which could position ambrisentan as the treatment of choice for PAH and potentially other cardiovascular disorders.

## A Word from the Physician



"The course of PAH is often one of steady deterioration and reduced life expectancy. Early detection and appropriate treatment of PAH may significantly improve patients' lives and increase survival; therefore the primary goals of PAH treatment are to improve symptoms, including increasing a patient's exercise capacity, leading to improved quality of life and a better chance of survival."

—Lewis J. Rubin, M.D.,

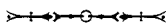
*Professor of Medicine, Director, Pulmonary Vascular Center, University of California, San Diego*



## RESISTANT SYSTOLIC HYPERTENSION

Hypertension affects approximately 50 million individuals in the United States and approximately one billion worldwide. Untreated high blood pressure greatly increases the chance of heart attack, stroke and the progression of chronic kidney disease. Despite the availability and use of several classes of drugs to treat hypertension, a significant percentage of these patients do not achieve blood pressures within the recommended range, a condition referred to as "resistant hypertension."

## Resistant Systolic Hypertension



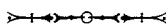
The "Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure" (JNC7) defines resistant hypertension as "the failure to achieve goal blood pressure in patients who are adhering to full doses of an appropriate three-drug regimen that includes a diuretic."

According to JNC7, a systolic blood pressure of less than 140 mmHg and a diastolic blood pressure of less than 90 mmHg are recommended for patients with hypertension and no other serious conditions. For patients with serious conditions, such as diabetes and chronic kidney disease, target systolic and diastolic blood pressures are more stringent—systolic of less than 130 mmHg and diastolic of less than 80 mmHg.

Clinical studies in hypertension have shown that diastolic blood pressure can be controlled to a goal of

90 mmHg in approximately 90% of hypertensive patients. However, in these same studies, guideline-recommended goals for systolic blood pressure were achieved in only 60% of patients, even when multi-drug regimens were utilized. Clinical studies have also shown that hypertension in patients with diabetes or chronic kidney disease is consistently more difficult to manage, requiring treatment with a multi-drug regimen. Despite intensive, multi-drug therapy, however, only 50% of these patients reach standard blood pressure goals, with even fewer reaching the more stringent blood pressure goals now recommended by JNC7. Moreover, data from a recent study conducted in a hypertension specialist clinic revealed that more than half of the diabetic patients examined required treatment with three or more antihypertensive drugs and only 22% of the patients studied achieved systolic blood pressure of less than 130 mmHg.

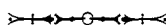
## What Is Myogen Doing?



We believe a considerable number of individuals with hypertension, especially those with diabetes or chronic kidney disease, are at risk for significant progressive cardiovascular and renal complications due primarily to elevated systolic blood pressure that is resistant to currently available therapies. As a result, we believe that there is a significant opportunity for a drug that is capable of lowering blood pressure in this resistant patient population, leading to the potential for enhanced patient outcomes. Darusentan, like

ambisentan, is an  $ET_A$  selective endothelin receptor antagonist (ERA) and has demonstrated a significant anti-hypertensive effect in patients with moderate essential hypertension. As an ERA, darusentan affects blood pressure by a mechanism different than other approved antihypertensive drugs. We are currently conducting a Phase 2b clinical evaluation of darusentan in patients with resistant systolic hypertension. We believe darusentan has the potential to be the first ERA available for treatment of resistant hypertension.

## A Word from the Physician



"Individuals with poorly controlled blood pressure are at significant risk for cardiovascular and cerebrovascular morbidity and mortality and represent a potentially substantial burden to the healthcare system. Setting appropriate blood pressure goals and working to meet them through aggressive anti-hypertensive treatment, with multiple agents, can reduce those risks."

—Henry R. Black, M.D.,

Charles J. and Margaret Roberts Professor and

Chairman Department of Preventive Medicine, Rush University Medical Center

## 2005 Milestones



- Report ESSENTIAL I & II preliminary results in the middle of the year;
- Complete patient enrollment in ARIES-2 by the end of June;
- Complete the darusentan Phase 2b trial in resistant systolic hypertension in the middle of the year;
- Complete patient enrollment in ARIES-1 during the fourth quarter; and
- Report ARIES-2 preliminary results by the end of the year.

## Management Team



J. William Freytag  
Ph.D.



Michael R. Bristow  
M.D., Ph.D.



Michael Gerber  
M.D.



Richard Gorczynski  
Ph.D.



John Julian

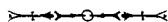


Joseph Turner



Andrew Dickinson

## Corporate Information



### Board of Directors

J. William Freytag, Ph.D.  
*Chairman, President  
and Chief Executive Officer*

Michael R. Bristow, M.D., Ph.D.  
*Chief Science and Medical Officer*

Kirk K. Calhoun  
*Chairman—Audit Committee*

Jerry T. Jackson  
*Chairman—Nominating and  
Corporate Governance Committee*

Daniel J. Mitchell  
*Sequel Venture Partners*

Arnold L. Oronsky, Ph.D.  
*InterWest Partners*

Michael J. Valentino  
*Adams Respiratory Therapeutics*

Sigrid Van Bladel, Ph.D.  
*New Enterprise Associates*

### Management Team

J. William Freytag, Ph.D.  
*Chairman, President  
and Chief Executive Officer*

Michael Bristow, M.D., Ph.D.  
*Chief Science and Medical Officer*

Michael Gerber, M.D.  
*Senior Vice President, Clinical &  
Regulatory Affairs*

Richard Gorczynski, Ph.D.  
*Senior Vice President,  
Research & Development*

John Julian  
*Senior Vice President,  
Commercial Development*

Joseph Turner  
*Senior Vice President,  
Finance & Administration  
and Chief Financial Officer*

Andrew Dickinson  
*Vice President, General Counsel  
and Secretary*

**Independent Auditors**  
Ernst & Young LLP  
Denver, Colorado

**Legal Counsel**  
Cooley Godward, LLP  
Broomfield, Colorado

**Corporate Headquarters**  
7575 W. 103rd Avenue  
Suite 102  
Westminster, CO 80021  
303.410.6666

**Transfer Agent  
and Registrar**  
Communications concerning  
stock transfer requirements,  
lost certificates and change of  
address should be directed to  
Computershare Trust Company, Inc.,  
350 Indiana Street, Suite 800,  
Golden, CO 80401, 303.262.0600,  
[www.computershare.com](http://www.computershare.com).

### Stockholder Inquiries

Inquiries from our stockholders  
and potential investors regarding  
our Company are always welcome.  
Please direct your requests for  
information to:

Derek Cole  
Director, Investor Relations  
Myogen, Inc.  
7575 W. 103rd Avenue  
Suite 102  
Westminster, Colorado 80021 USA  
303.410.6666  
[Derek.cole@myogen.com](mailto:Derek.cole@myogen.com)

### Website

[www.myogen.com](http://www.myogen.com)

### Stock Listing

Myogen, Inc. common stock is  
listed on the Nasdaq National  
Market under the ticker symbol  
MYOG.

### Annual Meeting

The next annual meeting of  
stockholders will be held  
on May 11, 2005 at 9:00 a.m.  
(Mountain) at The Westin  
Westminster, 10600 Westminster  
Boulevard, Westminster,  
Colorado.

**SAFE HARBOR STATEMENT** This annual report contains forward-looking statements that involve significant risks and uncertainties, including the statements relating to reporting of preliminary results from the Company's pivotal Phase 3 trials of enoximone capsules and ambrisentan, completion of patient enrollment in the Company's pivotal Phase 3 trials of ambrisentan and completion of the Company's Phase 2b trial of darusentan. Actual results could differ materially from those projected and the Company cautions investors not to place undue reliance on the forward-looking statements contained in this report.

Among other things, the Company's results may be affected by its effectiveness at managing its financial resources, its ability to successfully develop and market its current products, difficulties or delays in its clinical trials, difficulties or delays in manufacturing its products, and regulatory developments involving current and future products. Delays in clinical trials, whether caused by competition, adverse events, patient enrollment rates, regulatory issues or other factors, could adversely affect the Company's financial position and prospects. Results from earlier clinical trials are not necessarily predictive of future clinical results. Preliminary results may not be confirmed upon full analysis of the detailed results of a trial. If the Company's product candidates do not meet the safety or efficacy endpoints in clinical evaluations, they will not receive regulatory approval and the Company will not be able to market them. Even if the Company's product candidates meet safety and efficacy endpoints, regulatory authorities may not approve them, or the Company may face post-approval problems that require the withdrawal of its product from the market. If our discovery research program is not successful, we may be unable to develop additional product candidates. If the Company is unable to raise additional capital when required or on acceptable terms, it may have to significantly delay, scale back or discontinue one or more of its drug development or discovery research programs. Myogen is at an early stage of development and may not ever have any products that generate significant revenue.

Additional risks and uncertainties relating to the Company and its business can be found in the "Risk Factors" section of Myogen's Form 10-K for the year ended December 31, 2004 and Myogen's periodic reports on Form 10-Q and Form 8-K. Myogen is providing the information contained in this report as of the date of the report and does not undertake any obligation to update any forward-looking statements as a result of new information, future events or otherwise.



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